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## Can Patients With Active Duodenal Ulcer Fast Ramadan?

TO THE EDITOR: During Ramadan (ninth month of Hijra calendar), Muslims all over the world abstain from food and drink from dawn to dusk. This change of eating pattern during this month exerts effects on various physiological parameters (1, 2), including gastric acid secretion or gastrointestinal motility (3, 4). Although Allah has made fasting obligatory upon every Muslim, He has exempted those in whom fasting or any other illness may aggravate their conditions. Therefore, it is usual for Muslim patients, having gastrointestinal disturbances, especially peptic ulcer, to ask about the effects of fasting on their gastrointestinal condition. An epidemiological study has shown an increase in digestive disturbance during the first week of Ramadan (5). However, reports about the effects of Ramadan fasting on active peptic ulcers of patients on medical treatment are scant. A clinical study was, therefore, designed to assess the effect of Ramadan fasting on the healing process of peptic ulcer of patients on omeprazole (a proton pump inhibitor) treatment.

Fifty patients were selected, by simple sampling, having one or more of the following criteria, confirmed in endoscopy:

1. Active duodenal ulcer;
2. Multiple superficial bulbar ulcers;
3. Pyloric channel ulcer;
4. Active gastric ulcer;
5. Erosive duodenitis;
6. Duodenal or gastric ulcer with active bleeding.

All patients received 20 mg of omeprazole *b.i.d.*, except the ones with a positive urease test who received amoxicillin (1000 mg) and metronidazole (500 mg) (*b.i.d.*, for 2 wk), other than omeprazole. Patients were free to fast or break it. Eighteen patients observed the fast, and 21 patients broke it. Then, during 5 days, from the end of Ramadan, patients were endoscopied again and the results compared with the endoscopic findings of these patients at the beginning of the study.

Thirty-nine patients with duodenal ulcer completed the study. Among them, 18 patients observed the fast for 25–30 days, whereas 21 patients broke it. Two fast and two nonfast patients were smokers. None of the patients consumed nonsteroidal anti-inflammatory drugs or alcohol during the study. Statistically, there were no differences between the ages or sexes in the two groups. Endoscopic findings documented a 94.4% (17 of 18) cure in the fast group and a 95.5% (20 of 21) cure in the nonfast group. In an endoscopic picture of one patient in the fast group, multiple superficial bulbar ulcers could be observed. He had observed the fast for the first and second days of Ramadan, but had to break it afterward because of severe epigastric pain. The cure was not more than 10% at the end of the study. One patient in the nonfast group was a 45-yr old smoker. In his endoscopic picture, at the beginning of the study, an active duodenal ulcer without deformity could be observed. Not more than 50% improvement could be observed despite taking his drugs thoroughly. Other patients did not have any complaints throughout the study and even 3 months later, on follow-up.

The current study demonstrated that people could fast even with having active doudenal ulcer if they took the drug regimen employed in this study. Furthermore, fasting does not have a deteriorating effect on healing of duodenal ulcer. The results of this study are in contrast with the suggestion of Feldman *et al.* (6) who claimed that patients who suffer from acute duodenal or gastric ulcers should not fast, and asymptomatic patients might try fasting, if they could take a histamine-2 blocker at Iftar or Sahur (6). Our data are also in contrast with the suggestion of Azizi (5) who claimed that patients with active duodenal ulcers should not fast, even the ones on treatment. It should be noted that the drug regimen employed in this study is different from the one (cimethidine) employed in the Azizi's report (5).

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## First Reported Case of Colitis Cystica Profunda in Association With Crohn's Disease

TO THE EDITOR: Colitis cystica profunda (CCP) is an uncommon condition characterized by benign, mucus-filled cystic lesions within the submucosa. Histologically, it can mimic invasive adenocarcinoma.

A 37-yr-old white man was admitted to our hospital with a 1-day history of bloody diarrhea, crampy lower abdominal pain, nausea, and bilious emesis. Past medical history was significant for multiple sclerosis, which was currently in remission. He denied taking any prescription or over-the-counter medications. There was no family history of any colorectal diseases.

Physical examination revealed a well-developed man in moderate distress caused by abdominal pain. His vital signs were: temperature 40°C, pulse 132/min, blood pressure 118/70 mm Hg, respiratory rate 18/min. The abdomen was distended and diffusely tender to palpation. Bowel sounds were hyperactive. Peritoneal signs were absent. Digital rectal examination disclosed a scant amount of blood-tinged stool. The rest of the physical examination was unremarkable.

Blood counts showed Hb of 16.5 g/dL and a white blood cell count of 21 k with 28% bands. Serum electrolytes, liver and pancreatic enzymes, urinalysis, as well as stool studies were unremarkable. An abdominal CT scan revealed diffuse colonic wall thickening. A flexible sigmoidoscopy showed mild diverticulosis, apthous ulcers, and severe edema, which prevented advancement of the endoscope beyond 25 cm from anal verge.

Colonic biopsies showed intact crypt architecture without neutrophilic infiltrates, cryptitis, or crypt abscesses. Treatment with *i.v.* ciprofloxacin and metronidazole did not result in improvement. Repeat sigmoidoscopy performed 1 wk later showed similar endoscopic and biopsy findings. The patient was then started on corticosteroids for possible inflammatory bowel disease.

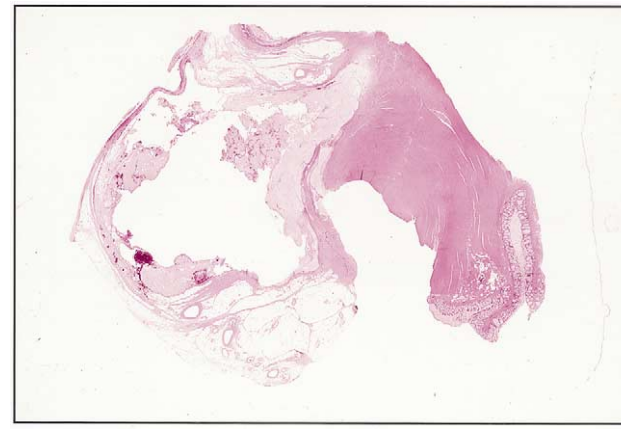

**Figure 1.** CCP: Submucosal mucus-filled cystic lesion.

On hospital day 15, his symptoms worsened. A CT scan of the abdomen showed colonic obstruction. Urgent surgery demonstrated a sigmoid stricture with fat creeping of serosa. Sigmoidectomy and end colostomy were performed. Gross pathology of the resected colon revealed surface adhesions and a lumen diameter of 0.7–1 cm. In the area of the stricture, a 1.5–cm sac-like space was identified adjacent to the bowel wall, which was filled with mucoid-appearing material. Histologically, there were multiple cystic-appearing lesions in the muscularis propria filled with acellular mucin (Fig. 1).

Subsequent colonoscopy through colostomy revealed extensive patchy ulceration of the distal transverse and descending colon. Biopsies demonstrated focal clusters of multinucleated histiocytic giant cells, crypt distortion, and active mucosal inflammation with increased mononuclear cells and neutrophils in the lamina propria along with mucosal ulceration consistent with Crohn's disease. Treatment with infliximab resulted in prompt remission.

CCP is an uncommon condition, which has been reported in association with solitary rectal ulcer syndrome, rectal prolapse, adenomatous polyps, radiation enteritis, and ulcerative colitis. Most people believe that it is postinflammatory or post-traumatic in origin (1). However, the rarity of this condition suggests that some unknown mechanism may play an additional role in pathogenesis. It typically presents with passage of blood and mucus in stools but can also present as diarrhea and abdominal pain. Barium enema studies demonstrate filling defects or thickened mucosal folds, which are often interpreted as mass lesion (2). Endoscopically, CCP appears as thickened edematous bowel wall or sessile polypoidal mucosal lesions. Endoscopic ultrasound shows the presence of large submucosal cystic lesions.

Although CCP has not been described in association with Crohn's disease, there have been few case reports of "*En-*